THE DIVERSITY OF TRIMETHOPRIM RESISTANCE WITHIN HOSPITAL ISOLATES

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Trimethoprim resistance within Enterobacteria can be determined either by the chromosome or by Resistance plasmids (R-plasmids). Epidemics of trimethoprim resistance have been caused by the spread of trimethoprim R-plasmids within the population (Grüneberg and Bendall 1979; Amyes et al 1980). Recently, however, transposons carrying trimethoprim resistance have been found to reside not only on R-plasmids but also on the chromosome of clinical strains (Towner et al 1982).

Routine isolation of strains from Edenhall Hospital in Musselburgh has revealed that a surge of trimethoprim resistance was occurring within the Gram-negative rods isolated from urine specimens. Of these strains, an unprecedented 64% were resistant to 10 mgL^{-1} trimethoprim and 25.5% to 1 gL^{-1} trimethoprim. One hundred isolates were examined in detail to determine which type of trimethoprim resistance was being observed. Fifteen strains were shown to possess auto-transferable trimethoprim resistance and examination of the plasmids by their drug resistance pattern and DNA size revealed that there was a minimum of six different types. Nine strains were Pseudomonas aeruginosa and although some of these showed a high degree of insusceptibility, no auto-transferable trimethoprim R-plasmids were shown. Four Proteus species were shown to possess low insusceptibility to trimethoprim but possessed auto-transferable R-plasmids. Within the Proteus strains these plasmids conferred a minimum inhibitory concentration (MIC) of trimethoprim of 100 mgL^{-1}. However, when these plasmids were transferred into the standard recipient strain, Escherichia coli $J6\text{-}2\text{-}_2$, they nevertheless elevated its MIC of trimethoprim to above 1 gL^{-1}.

The R-plasmids isolated from the Musselburgh clinical isolates were examined for the presence of transposons conferring trimethoprim resistance. It was found that there was a predominance of transposon 7, the 8.7 Mdal plasmid that determines trimethoprim and streptomycin resistance. The four strains that possessed high level resistance, but were unable to transfer trimethoprim resistance, were examined for the presence of transposons within the bacterial chromosome. Transposon 7 was found within the chromosome of some Enterobacteria strains but could not be detected in the chromosome of any Pseudomonas strains.

The incidence of trimethoprim resistance was higher amongst the Gram-negative rods isolated in this hospital than in other hospitals in the district. This seems to reflect the nature of the patients from whom the strains were isolated. There were many types of trimethoprim-resistant organisms within this small population and the spread of one type of strain or R-plasmid was not being observed. The high incidence of trimethoprim resistance amongst these hospital isolates seems to be linked to the clinical use of ampicillin rather than trimethoprim and this supports the results we have found in other hospitals (Amyes et al 1980). The use of ampicillin in this hospital has now been restricted.

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